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EDITORIAL COMMENT

Dietary Research in Heart Failure

Beyond the Salt Shaker*

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Anyone caring for patients with heart failure (HF) is aware of the importance of dietary instruction for these patients. Because of the patients' salt-avid state, these instructions generally follow the admonition for a reduction of sodium intake. In a related fashion, particularly for patients with evidence of volume overload and diuretic therapy requirements, fluid restriction is the next most common instruction. Finally, often related to an atherogenic substrate, dietary restriction of saturated fats is commonly the third admonition. In general, aside from the dietary restrictions related to a patient's evidence of edema or the diagnosis of coronary artery disease or hyperlipidemia, little attention has been paid to dietary assessment, evaluation, instruction, or research for the vast majority of HF patients. This is, indeed, unfortunate because there are suggestions that as many as 50% of patients hospitalized with HF are malnourished (1).

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Heart failure textbooks and guidelines add little additional information or suggestion of the magnitude of the need for information regarding nutritional assessment of patients with HF (2–4). A single text contains one page out of 929 focusing only on cachexia (2). And among the paucity of data exists the widest spectrum of opinion. A recent Framingham analysis related increased body mass index (BMI) with worsened survival (5). Conversely, other studies have suggested that survival may in fact be better with higher BMIs or, in fact, with obesity (6,7).

Were a student or resident to ask me about information on the general nutritional status of patients with HF, my response would suggest that weight loss in advanced disease due to malabsorption and wastage of nutrients coupled with a decrease in appetite seen in the volume overloaded patient is common. I would also mumble something about the need to attain ideal body weight. But the response would lack critical data, conviction, or, more importantly, suggestions aimed at proper nutritional replacement and support.

Therefore, I welcome the paper by Aquilani et al. (8) in this issue of the *Journal*. In the background information for this study the authors rightfully explain, "little information exists regarding the nutritional adequacy and alimentary habits of patients with clinically stable chronic HF." The authors looked beyond simple morphometrics and performed a prospective analysis of nutritional balance. The authors studied carefully 57 non-obese, community-living patients mostly with mild to moderate HF. The population is not perfect for broader extrapolation; the patients were briefly hospitalized for transplant evaluation, and, despite their moderate functional class, many had exceedingly low peak oxygen uptake. Nonetheless, the only neurohormone measured was plasma norepinephrine and these levels were only modestly elevated. Importantly, the authors excluded patients taking beta-blockers, concerned that metabolic rate might be affected. The population therefore might best be described as having Stage C (4) disease on suboptimal therapy—akin to many general HF populations! Age-, BMI-, and activity-matched subjects served as a suitable control group.

More important, however, is the author's study of the calorie-nutrition balance (CNB), which reflects a daily homeostasis between calorie-nitrogen intake over excretion and its relationship to total energy expenditure. The methods seem adequate, although one might debate how resting energy expenditure (REE) is measured or calorie/nitrogen intake (based on seven-day food diaries) assessed; again not an important point to belabor.

The authors demonstrate a remarkable proportion of patients having a negative calorie balance (70.1%), negative nitrogen balance (59.6%), or negative combined CNB (40.3%). This occurred despite similar calorie and nitrogen intake, not only in HF patients but also in controls; only underweight controls took in fewer calories. The next important finding is that total energy expenditure was the same in HF patients and controls. Doing the math, one then readily understands the authors' conclusions—these patients simply do not have enough energy available for physical expenditure on activity, let alone stresses related to periods of decompensation, infection, or enhanced physical training.

The authors' work, however, (and that of the HF community) remains undone. No new information is offered as to how or why the nutritional balance is so poor in these patients. Presumed is a progressive change in dietary habits coupled with chronic malabsorption of macro and micronutrients. Much could be learned by studying CNB in patients earlier along the HF path (Stages A and B). Whether cytokine activity, drug effects on taste, or even subclinical depression alters intake is not well known. Also, we do not know what forces may be at play to keep the energy expenditure of the "sedentary" HF patient so high. We also do not know what role the negative balance plays in the symptoms of the HF syndrome nor on its progression. And finally, we do not know the impact of prescribed weight loss or exercise training on these patients.

What we do now know is that our patients are likely starving before us, and we may be inadvertently making it worse. Unfortunately, calculating CNB is not a simple

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bedside procedure. Also, nutrition in HF patients is not likely to either share the exciting spotlight of new drug or device development in HF or have similar access to funding resources. Yet, many questions remain—questions that may play an important role in limiting our HF epidemic. We should applaud the authors on this novel approach. I also encourage young investigators, and all of us, to begin to think "beyond the salt shaker."

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